RESTRICTED

THE SCOPE OF PATHOLOGY IN THE GERMAN WEHRMACHT

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COMBINED INTELLIGENCE OBJECTIVES SUB-COMMITTEE

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Medical

COMBINED INTELLIGENCE OBJECTIVES SUB-COMMITTEE
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ORGANIZATION

1. The chief consultant in pathology to the Wehrmacht had the rank of colonel and was stationed in Berlin at the Heeres Sanitätsinspektion (comparable to the U. S. Surgeon General's Office). During the war this post was first held by Schürrmann, later killed on the eastern front and replaced by Lauche, who was succeeded by Randerath.

The chief pathologist was also director of the Institut für Allgemeine Wehrpathologie der Militärärztlichen Akademie in Berlin. In this institute were kept the protocols of all autopsies performed in the Wehrmacht. Gross specimens or histological preparations were sent there only in exceptional cases or when requested for special study.

The director had two assistants, one was entrusted with the classification and arrangement of the protocols; the other was curator of the pathologic anatomical collection, which included specimens from World War I. In 1943 a part of the staff as well as some of the protocols were moved to Giessen and housed in the department of pathology of the University of Giessen. In the last months of the war the anatomical collection was also moved from Berlin and transported to Mertschütz in Lower Silesia.

Unlike the United States Army Institute of Pathology in Washington, that of the Wehrmacht did not receive protocols and specimens of surgically removed tissues, nor did it receive the tissues and histological preparations of most autopsies. This material was allowed to remain in the possession of the local pathologists, and any more extensive studies were likewise carried out by them. Oberstabsarzt Lauche declared that this system proved less satisfactory than the centralization carried out in the U. S. Early in the war an effort was made to centralize the work in pathology, but id did not receive the support of the Sanitätsinspekteur.

2. In the Zone of the Interior pathologists were distributed as follows:

a. One consultant in pathology to the chief surgeon of each Wehrkreis (comparable to a service command). These men were usually the heads of the departments of pathology at the various universities.

b. One pathologist at each of the larger general hospitals. For this task men were chosen who were professors of pathology at universities or directed the laboratory in a

city hospital.

c. Pathological institutes belonging to the Wehrmacht and existing prior to the war. There were five of these located respectively in Königsberg, Berlin, Breslau, Hamburg, and Nürnberg. During the war the regular army officers were replaced by pathologists from the universities.

3. Distribution of pathologists in theaters of operation:

a. One consultant in pathology for each army group--usually a university professor or director of a large city hospital laboratory.

b. One consultant in pathology for each army--a university professor or chief of a civilian hospital lab.

c. One pathologist at each of the larger general hospitals--recruited from among the assistant professors at universities or the younger prosectors in civilian laboratories.

4. Any of the installations listed in paragraphs 2 and 3 could act as histopathological centers. A rigid channel for forwarding material from lower to higher echelons was not established; specimens were gathered and stored at any station having a pathologist. Only when special studies were being conducted were the smaller units required to forward the material for further study. However, copies of all autopsy protocols were sent to the consultant in pathology in Berlin.

5. The Luftwaffe had its own pathologists, headed by Oberstabsarzt Büchner, director of the Luftfahrt-Pathologisches Institut at the University of Freiburg. The German Navy had only two pathologists; one, working at Kiel, was responsible to the Baltic Fleet; the other, stationed at Wilhelmshaven, was the pathologist for the North Sea Fleet.

EQUIPMENT

1. Histopathological laboratories in the Zone of the Interior were an integral part of university institutes of pathology the director often working in both his civilian and military capacity. The larger laboratories in theaters of operations were usually housed in institutions belonging to the conquered countries.

2. The basic pathological laboratory equipment for the military general hospital, whether in Germany or elsewhere, consisted of three large chests. When emptied and properly arranged, these chests formed a work bench with

eight large drawers and ample desk space. Chest No. 1 contained equipment for histological studies; freezing microtome, 4 kgm cylinder of carbon dioxide, Leica photomitrographic unit, microscope, slides, dyes. Chest No. 2 held apparatus for the performance of autopsies; a dish for organs, weight scale, rubber apron, rubber gloves, mailing containers, and a case of instruments. The latter is an easily transportable wooden satchel which was produced in great numbers and was sent to even very small installations in order that autopsies could be performed there when necessary. It contained an adequate collection of knives, scissors, and forceps; a chisel, mallet, saw, rachitome, sounds, twine, needles; as well as pencil and paper. Chest No. 3 held several large mailing containers as well as quantities of formalin, Karlsbad salts, and chloralhydrate. The combined weight of the chests was 360 kg.

SPECIAL PATHOLOGICAL STUDIES

During the war various diseases were made the subject of investigations by competent pathologists. At the annual meetings of the consultant physicians of the Sanitätsinspektion the results of these studies were presented. The reports were incorporated in the published proceedings of the meetings, the "Bericht ueber die Arbeitstagung Ost der Beratenden Fachärzte". Only a very small number of copies were printed as they were semi-secret and were distributed primarily to those who took part in the meetings.

Below are abstracts of the findings of various investigators, based upon their reports in the "Berichte" and verbal communications to the writer.

Typhus

Oberarzt Dozent Schulze:

About 30% of all typhus patients, among them physicians and nurses, insist that they were never infested with lice. In one typhus lazarett for Russian prisoners of war the physicians and attendants wore special tight-fitting suits and rubber gloves. In spite of these precautions the majority contracted the disease, with the exception of those who wore Russian gas masks while in the infected wards. The infectious material rarely gains entrance through a skin abrasion, most often it follows contact with a mucuous membrane, particularly the conjunctiva. This may account for the frequency of conjunctivitis

among the technicians in the typhus laboratories of Cracow and Lemberg. The possibility of droplet infection due to coughing by typhus patients must also be considered.

Stabarzt Prof. Brinkmann:

The rickettsia lodge in the endothelial cells of the capillaries and pre-capillaries of the brain; the cells become swollen, and the vessel wall becomes hyaline and necrotic. Only short sections of the vessel are involved, and these are surrounded by a fibrinous exudate containing lymphocytes, plasma cells, and fibroblasts. This forms a spindle shaped lesion, the typhus nodule, which is associated with foci of glial proliferation, edema of the brain and meninges, and hemorrhages. Most frequently involved are the brain stem, floor of the 4th ventricle, and the corpus callosum. The typhus lesion is basically an arteriolitis comparable to periarteritis nodosa.

The thromboses and hemorrhages that occur occasionally lead to hemiplegia. The cranial nerves that are most frequently affected are the optic, facial, and acoustic. Their involvement accounts for the subjective clinical phenomena of vertigo, deafness, and tinnitus. The peripheral nerves may likewise be involved. In the lower extremities this often manifests itself as a polyneuritis; in the arms paralyses are more common, especially paralysis of the ulnar nerve. The predilection for the floor of the 4th ventricle explains the multiple vegetative disturbances--lowered systolic pressure, decubitus ulcers, trophic ulcers with gangrene of fingers and toes, as well as necrosis of the cartilages of the ear, nose, and larynx.

Oberstabsarzt Prof. Gruber (Göttingen):

The following findings are based on 65 autopsies and an additional 17 hearts of patients who died of typhus fever. No important differences were noted in the histopathology of patients that died of typhus during 1914-18 and those that died on the eastern front during the winter 1941-42. Hueck has recently pointed out that in addition to the usual typhus nodule there are lesions that consist chiefly of proliferating adventitial cells. These lesions are associated with little exudate, almost no injury to the vessel wall, and resemble granulomata. According to Gruber these atypical nodules substantiate his contention that even the more common type of perivascular typhus node should not be likened to the lesions of periarteritis nodosa. The skin lesions also often consist merely of a diffuse, non-specific lymphocytic

infiltrate about the smallest vessels of the corium. The typical nodules may be absent, making the skin biopsy of little diagnostic value.

Although typhus nodules are present in the heart musculature, they are not as compact as those found in the brain and may even be wholly absent. On the other hand, a diffuse interstitial myocarditis is always present. The infiltrates, consisting of lymphocytes and plasma cells, follow the course of the capillaries between the muscle bundles. More clearly circumscribed, even nodular lesions, were often found in the sub-pericardial and sub-endocardial connective tissue; e.g., in the tip of a papillary muscle. Degeneration and necrosis of the muscle fibers, such as occur in diphtheria, were not seen.

It should be emphasized that beside the typical perivascular typhus nodule there is a less characteristic loose lymphoctic infiltrate in the connective tissue of various organs.

Oberfeldarzt Prof. Lauche (Frankfurt):

In order to insure a definite histopathological diagnosis of typhus at autopsy the floor of the 4th ventricle, particularly in the region of the olive, should be examined; likewise the heart muscle. The skin lesions at the time of death are usually no longer sufficiently characteristic of typhus fever to permit diagnosis, or have disappeared. The formation of glia nodules in the brain of typhus patients begins during the first week of illness; in the third week, especially from the 16th to 18th days, the process reaches its height. During the 4th week there is a reduction in the number of nodules, though they persist into the 5th and 6th weeks. In the medulla the process is apparent on the 5th or 6th day of the disease, it persists longer than in the cerebral cortex, and is still active during the 5th and 6th weeks. Identical nodules are found in the central nervous system of cases of St. Louis encephalitis, encephalitis of Japan, and the endemic encephalitis described by Pette.

Stabsarzt Doz. Bohne (Hamburg):

Among 51 autopsied cases of typhus, 13 died of an acute cardio-vascular collapse (on 9-14th day of illness); 11 of lobar pneumonia (10 -28th day); 13 of broncho-pneumonia (8-23rd day); 4 of an acute diffuse hemorrhagic glomerulonephritis (11-41st day); 5 of generalized infections (19-32nd day); 3 of pharyngeal or laryngeal

diphtheria (17-40th day); 1 of ascending pyelonephritis (41st day); 1 of suppurative meningitis following otitis media (42nd day).

An acute hemorrhagic glomerulonephritis was found in 15 of 60 typhus autopsies; in only two cases had the diagnosis been made clinically. Histological examination showed albumen, erythrocytes, and leukocytes in the space of Bowman's capsule; plugs of white blood cells and red blood cells were present in the tubules; a massive infiltrate surrounded the glomeruli. Associated with the lesions of the renal parenchyma were petechiae and hemorrhages in the mucosa of the renal pelvis and bladder.

Evidence of hemolytic jaundice was found in 22 of 47 cases. Histologically there was hemosiderosis of the spleen, liver, bone marrow, and kidneys. Hemoglobinuric nephrosis with hemoglobin casts in the tubules was found in cases dying on the 10-14th day. Bile plugged the canaliculi of the liver; clinically the Van den Bergh test was indirect, blood bilirubin negative, and urine urobilinogen positive.

In 39 cases the posterior portion of the eyeballs was carefully examined. A dense lymphocytic (?) infiltrate was present in the supporting tissue of the optic nerve associated with a variable degree of gliosis. In the leptomeninges surrounding the optic nerve there were small perivascular infiltrates of lymphocytes associated with a slight endothelial proliferation. A rather loose lymphocytic infiltrate also surrounded the greatly dilated capillaries of the choroid. In the retina there were isolated and well circumscribed infiltrates of lymphocytes or histiocytes.

Wolhynian Fever

This disease, also known as trench fever or 5-day fever, affected over 10% of the troops in many regions of the eastern front during the winter 1941-42. The Russians were apparently singularly immune. The onset is often acute with high fever and chills. Malaise, headache, vertigo, and recurrent bouts of fever with pain in the muscles, bones, and joints, often associated with paresthesia, is the usual clinical picture. Recovery is usually complete after 1-3 weeks. However, Oberstabsarzt Schaltenbrand (Würzburg) calls attention to the frequency of polyneuritis in this disease. The neuritis begins when the fever is at its height, most frequently involves the legs, and may

resemble a Landry's paralysis. This may not clear up for over a year. Sensory disturbances are most marked in the lumbar rather than in the sacral segments.

Oberarzt Dozent Schulze:

The etiologic agent of Wolhynian fever is Rickettsia quintana, transmitted by the body louse. Unlike R. prowazeki of typhus fever, which is only attached to the surface of the intestinal epithelium of the louse, R. quintana penetrates the cells themselves. Since the organism is present in the circulating blood of the patients (as late as the 23rd day) it has been found possible to demonstrate its presence by allowing non-infected lice to feed on the patient. After several days the lice are killed and the intestinal tract examined for the presence of R. quintana. A disadvantage of this test is the fact that R. quintana is morphologically and culturally identical with R. pediculi; by repeated animal passage the latter may show the same pathogenicity as the former. Neither agglutinins nor precipitins can be demonstrated in the blood of patients from whom the rickettsia were obtained.

Stabsarzt Unger:

After examining thousands of thick blood smears stained with Giemsa, Unger is of the opinion that in over 600 he was able to demonstrate the pleomorphic rickettsia of Wolhynian fever. In nearly all of 300 cases he claims to have demonstrated the organism in the circulating blood; similar success accompanied his study of the spinal fluid in these patients. Other investigators who examined his preparations were not convinced that what Unger interpreted as R. quintana were not merely non-specific granules.

Bacillary Dysentery

Oberstabsarzt Prof. Bohmig:

Although the etiology of dysentery is solved, its pathogenesis is still largely unknown. The pathologic-anatomical beginning of the disease is localized in the rectum, the specific changes are restricted to the intestinal mucosa, and the non-specific changes to the regional lymphnodes. The circulating blood and other organs remain wholly free of the organisms, as well as of any noteworthy reactions except for a variable leukocytosis and rare joint or nervous system manifestations.

Bacillary dysentery is preceded by a non-specific catarrhal inflammation of the gastrointestinal tract brought about by dietary indescretions, lowered body temperature, etc. If at this time dysentery bacilli are present in the intestine, infection may occur. The organisms first lodge in the region of the rectum, producing the earliest specific lesion in the form of a pseudomembrane. The explanation for this localization in the rectum may be sought in 1) the very slight resorptive and secretory capacity of the mucosa in this part of the tract; 2) intestinal spasms and mucosal defects. The spasms keep the feces in the rectum, sigmoid, and descending colon, furthering the attachment of dysentery bacilli in places where a microscopic loss of epithelial cells has occurred. They also account for the gradual retrograde extension of the specific lesion as far as the caecum, or even into the small intestine if the ileocaecal valve is insufficient.

The mucosal lesions are the result of an interplay between inflammatory processes and necrosis. In the first stage there is only an inflammatory reaction limited to the mucosa. In the second stage necrosis, likewise limited to the mucosa, becomes apparent. The effect of the bacterial toxins is evidenced by the absence of reaction on the part of the epithelium or connective tissue, by the lake-like dilatation of the capillaries, by the absence of a cellular exudate or thrombosis, and by the late appearance of a limiting inflammatory response in the submucosa. The third stage begins at the site of the lymph follicles. Here there is a physiological interruption of the muscularis mucosae, so that after necrosis and emptying of the follicle the infection can spread from its base into the submucosa. The toxic action of the dysentery bacillus is exhausted in the necrosis of the mucosa and lymph follicles. The ulceration which follows is non-specific. Pseudomembranes or fresh areas of necrosis, the specific tissue changes of dysentery, are absent from all older and larger ulcers. Regeneration of the epithelium occurs quickly, only to be destroyed by the non-specific inflammatory processes.

The chief characteristic of dysentery is that the bacilli are always limited to the intestine and regional lymphnodes. A bacillemia is absent. Nor is there a significant toxemia, for if there are the circulating blood and hematogram would be different and the regional lymphnodes would show characteristic lesions. As a specific response to the dysentery bacillus there remains only the desquamation associated with inflammation, the necrosis as the result of the effect of the bacillary toxins, and

perhaps the large shallow ulcerations. The formation and absorption of endotoxins is incomparably less than that occurring in diphtheria--a fact which may be related to the abundance of bacteriophages in the intestine. Only the rare cases of sudden death early in the course of the disease are attributable to the toxins, which produce a vasomotor paralysis. Much more common is death from exhaustion late in the illness due to loss of the mucosa of the large intestine.

Oberarzt Dozent Walther:

In 20 autopsies of patients who died of dysentery (bacillary) determinations were made of the water and chloride content of the skin, subcutaneous adipose tissue musculature, and liver. The water content of the skin averaged 60% in comparison with the normal 70%; the lowest values were slightly over 40%. In the subcutaneous adipose tissue values of 9-14% were found; the norm is 26-30%. If there had been a marked loss of fat the water content was considerably higher, but still less than normal. The skeletal muscles and internal organs such as the liver and kidney showed no loss of water.

The chloride deficit was even more pronounced. Instead of the usual 300 mg% Cl, the skin contained only 120-200 mg%. In the subcutaneous adipose tissue there was a comparable loss, and even the musculature showed a decreased chloride content.

In severe cases of bacillary dysentery, as in other infections, there is a disturbance of the third phase of blood coagulation. Determination of the thrombocyte count, thrombocyte agglutination, and the effect of the addition of thrombokinase and normal thrombocytes revealed that this phenomenon is not associated with a change in the platelets or the thrombokinase. Rather, it may be traced to a toxic alteration of the fibrinogen.

Amoebic Dysentery

Oberfeldarzt Prof. Rodenwaldt:

Although it is generally recognized that Entamoeba coli is present as a saprophyte in the intestinal tract of men, the pathogenicity of E. histolytica has never been doubted since the publication of Schaudinn's investigations. This belief is supported by the repeated appearance of erythrocytes in the cytoplasm of E. histolytica, the occurrence of this amoeba in the depths of the intestinal ulcers of

acute and chronic cases of tropical dysentery, and its presence in the pus of amoebic abscesses of the liver.

During the last war it came as a distinct surprise that the amoebae were found in the stools of returning soldiers who gave no history of dysentery. Subsequently the organism was found in about 14% of the civilian population (of Germany), none of whom had ever had nor subsequently developed the disease. In these cases the entamoebae contained no erythrocytes, but they were identical with E. histolytica. The cysts had four nuclei and were otherwise indistinguishable from those of the pathogenic form.

Recent investigations have shown that E. histolytica occurs in men in two forms: 1) the amoeba of the tissues, E. histolytica in the strict sense of the term; 2) the amoeba of the intestinal lumen, the minuta-variety of E. histolytica, a harmless saprophytic intestinal parasite. Most important, however, is the fact that the pathogenic phase of E. histolytica, the tissue parasite, never produces cysts. Only the minuta-variety has that ability. This leads to the important conclusion that even in the presence of a history of dysentery, the finding of cysts in the stool does not warrant the diagnosis of amoebic dysentery, for the dysentery may be due to other causes.

The only conclusion permissable after the finding of minuta forms in the stool is that the individual is a potential victim of amoebic dysentery. The present opinion is that the minuta variety may become the tissue form when some secondary factor has produced a local area of decreased resistance, thereby permitting the invasion of the intestinal mucosa by the amoebae. Such secondary factors occur more frequently in the tropics than in temperate climates. However, this concept is not accepted by all investigators. Thus, it is known that the per anum injection of E. histolytica into kittens leads to pathologic changes typical of amoebic dysentery. But Westphal showed that the same picture can be produced if a cell free filtrate of the infected stool is injected. May another agent, perhaps a virus, accompany the tissue form of E. histolytica and act as the primary causal agent of the disease? For practical purposes, of course, E. histolytica remains an important indicator of a specific intestinal disease which has been identified with the name "amoebic dysentery".

Malaria

During the war interest centered about the control of malaria among the troops, primarily by the use of prophylactic doses of atebrin. Histopathological studies of the disease have not been very extensive. Stabsarzt Horing (Tübingen) had extensive experience with the malaria problem in Greece. He believes that many cases of recurrence may be due to the persistence of sporozoites within the tissues following massive infections by one or more mosquitoes. Stabsarzt Rix (Nürnberg) has been studying the frequency of exo-erythrocytic development of the plasmodium, particularly in cells of the reticuloendothelial system. Like other investigators he has found that this process is uncommon and difficult to demonstrate. Exo-erythrocytic development can scarcely account for the great frequency of relapse in treated cases of malaria.

Diphtheria

Oberstabsarzt Prof. Voit:

Diphtheria was a source of serious concern to the medical corps on the eastern front, particularly during the winter of 1941-42. The disease was remarkable for the fact that the tonsils and pharynx frequently showed no pathological changes, not even hyperemia. Often the entire course of the illness would be manifested only by a high fever and severe hoarseness. The first few cases were diagnosed only at the post mortem examination; in fact, throughout the epidemic as many as 50-60% of the cases were not recognized before death. At atopsy the diphtheritic membrance extended far into the trachea and even into the bronchi. This localization and the associated high fever is uncommon in adults, being observed more frequently in children.

Post diphtheritic paralyses and myocardial damage were quite common; weakness and paralysis of the legs with severe gait disturbances were repeatedly observed. Early paralyses (2-3 weeks) involved the palatal and oculomotor muscles, peripheral motor paralysis, and Landry's ascending paralysis. Myocarditis, if it did not lead to sudden death, had a good prognosis; permanent myocardial damage was uncommon. The time required for healing varied from several weeks to 3-6 months.

Oberfeldarzt Prof. Burgers:

Wound diphtheria was not infrequent, but it showed

little relation to the incidence of pharyngeal or laryngeal diphtheria. However, in Bad Nauheim wound diphtheria appeared in three military hospitals and was traced to pharyngeal diphtheria (carriers?) in the nursing staff. The outcome of the disease was seldom fatal and complications such as neuritis or myocarditis were uncommon. Three types of wound diphtheria have been recognized: 1) Typical wound diphtheria. In this type the wound suddenly alters its appearance; the granulation tissue becomes very lax and flaccid; ulcers appear that have notched borders and undermined edges; and the surrounding tissues become hyperemic. Cultures yield Corynebacterium diphtheriae. 2) The type in which the wound is normal in appearance, but from which true diphtheria organisms are obtained in pure culture or mixed with pyogenic cocci. 3) Wounds which clinically resemble those of diphtheria, but from which no true C. diphtheriae can be cultured.

Since most laboratories use Loeffler's medium or the Clauberg plate with dextrose, the hyperacidic pseudo-diphtheriae have often been erroneously identified as C. diphtheriae. The differentiation of these two organisms is simple if the modified Clauberg plate (substitution of serum or ascitic fluid for blood) is used, in conjunction with the sucrose fermentation test. The intradermal inoculation of a guinea pig with true diphtheriae of the gravis or intermedius varieties gives rise to a small abscess and slight edema; the hyperacidic pseudodiphtheriae leads to a slight leukocytic infiltration without edema and never kills the animal.

Tularemia

In 1938 there was an epidemic of 5,000 cases of tularemia in the valley of the Volga River. When the Germans entered that region in 1941-42, they likewise suffered severely from the disease and reported several thousand cases. The disease was transmitted by mice, whose number had increased greatly when the harvested grain was left standing in shocks in the fields. Infection of the soldiers occurred primarily by ingestion of contaminated food or water, as well as by inhalation of dust particles of the dried mouse feces. The ulcero-glandular and oculo-glandular types of infection were very uncommon. The type most commonly observed presented the so-called "typhus picture" with apathy and malaise. These cases were frequently erroneously diagnosed as la Grippe rather than tularemia.

Oberfeldarzt Prof. Lauche (Frankfurt) carried out the histopathological studies. These revealed nothing that was

new. In this work he had the assistance of Dr. Reichle of the St. Katherine Hospital in Stuttgart. Dr. Reichle had studied the disease while serving as the pathologist at the municipal hospital in Cleveland, Ohio.

Epidemic Hepatitis

Oberfeldarzt Prof. Gutzeit:

The great epidemic of hepatitis which appeared in the autumn of 1941 was preceded by smaller ones in the fall of 1939 and 1940. Although the disease took its greatest toll among the troops on the eastern front, it also appeared among the civilian population of Germany. Beside the epidemic cases there were sporadic ones without a demonstrable chain of infection. If the etiology or pathogenesis of these sporadic cases differs from those occurring in epidemics, one would not expect that their incidence would increase during epidemics. However, a study of the sick reports in the Archiv für Wehrmedizin from 1 July 1939 to 30 June 1940, and again from 1 October 1940 to 31 December 1940, reveals a complete synchronization of the time - incidence of the epidemic and of the sporadic forms. Gutzeit accepts this as proof that the large majority of sporadic cases of epidemic (contagious) hepatitis are identical with those occurring in epidemics. All efforts to find clinical differences between the two varieties have failed.

The frequency of liver damage following the use of arsenicals in the treatment of syphilis increased suddenly with the rising incidence of epidemic hepatitis. A similar relationship was noted in World War I and Gutzeit suggests that many of these cases of liver injury after arsenic therapy are in part due to the presence of the virus of epidemic hepatitis.

Drs. H. Axenfeld & K. Brass:

These investigators studied 27 cases of epidemic hepatitis by means of biopsies obtained during the first 120 days after onset of jaundice (Frankf. Ztschrft. f. Path. 57: 147-256, 1942 and 58: 220-238, 1944). Ten were sporadic cases and 17 occurred during an epidemic; no differences could be found in the histopathology of the two types. The early stages showed, 1) degenerative changes in the liver cells which usually progressed to a more or less widespread central necrosis, and 2) marked activity of the reticuloendothelial apparatus. While parts of the parenchyma were still being destroyed, regeneration

was taking place in other areas, as evidenced by mitotic and later amitotic division of the liver cells.

Local vascular disturbances apparently played an important role early in the disease; the central portions of the lobules being the chief site of parenchymal destruction. The mesenchymal interstitial tissues were spared, though occasionally slight changes were noted. The subacute and subchronic stages were identical with what Eppinger has called the "periacinöse" form of catarrhal jaundice. It is now clear that the latter is not a separate disease entity but is merely the subacute stage of epidemic hepatitis. Furthermore, when all anatomic findings are reviewed, it is evident that nowhere is there evidence against the assumption that epidemic hepatitis and catarrhal jaundice are one and the same disease.

After the acute phase had passed, the reticulo-endothelial reaction regressed - the intralobular more quickly than the interlobular. Healing, in the anatomical sense of the word, did not set in for a considerable period after clinical jaundice had disappeared. In the majority of cases there was eventually complete restitutio ad integrum; in some a residual central sclerosis remained. In a small percentage this became progressive, suggesting transformation into cirrhosis.

Stabsarzt Doz. Dohmen:

It may be assumed that if an infectious agent is the cause of epidemic hepatitis it can be found in greatest concentration in the liver of patients ill with the disease. Bits of such livers were removed as biopsies and inoculated into various animals and into chick embryos. In the course of these experiments it was shown that white mice became ill after inoculation with human infected liver. Material from 13 of 16 human biopsies produced pulmonary and hepatic lesions in the inoculated mice. Seven of the 13 strains were carried through many (12-32) animal passages. The following description is based upon results obtained with 1900 mice.

After inoculation with the initial biopsy material, the animals became ill within 14-21 days; lesions were demonstrable in the lungs, liver, and spleen. The gross changes in the lung were identified as hemorrhages after histologic examination. The liver and spleen were always enlarged, the spleen being as much as 8 times normal size. Multiple small white discolorations were often found beneath

the capsule of the liver; jaundice was absent. Small hemorrhages were occasionally found in organs other than the lung and were interpreted as a part of the hemorrhagic diathesis.

Histologically the hepatic lesions were characterized by maximal dilatation of the capillaries. The latter were often filled with an albuminoid amorphous mass and contained large numbers of desquamated endothelial cells. The Kupfer cells were increased in number. In the liver cells themselves often were extensive cytoplasmic and nuclear changes associated with increased size of the cells. The nuclei stained poorly and were pyknotic and fragmented; the cytoplasm was pale and hydropic and only a few strands appeared to surround the nucleus. The enlargement of the liver cells led to their partial displacement and resulted in alteration of the lobular architecture. These changes resembled those identified by Voegt in his human biopsy material as capillaritis with secondary hepatosis. Histological examination of the surface discolorations noted grossly revealed a necrosis similar to that seen in acute yellow atrophy.

The period of incubation, which at first lasted 2-3 weeks, was reduced to 3-6 days after repeated animal passage. The animals became acutely ill and nearly all died. The disease developed after subcutaneous, intrapulmonary, intracerebral, or intraperitoneal injection of the ground up liver or lung from an infected animal. Cell free filtrates of this material produced similar changes when injected into mice.

Incubated chicken eggs were likewise inoculated with human biopsy material. This produced hyperemia of the allentoic sac, but the fluid in the latter remained clear. The embryos did not die. This was in contrast to the results obtained by Siede and Lutz of Leipzig who inoculated fertile hen's eggs with duodenal fluid from cases of epidemic hepatitis. After the 4th egg passage mice were inoculated with the material: they all died and showed gross and microscopic lesions identical with those obtained by the primary inoculation of human biopsy material. After the 8th animal passage chick embryos were again inoculated. After one chick passage, mice were again injected for an additional six passages. In all cases the same changes were observed, and Dohmen concludes that the etiologic agent of epidemic hepatitis is a filtrable virus.

Neutralization experiments were carried out on 252 mice. Triturated liver and lungs of typically diseased animals were incubated for two hours with serum from patients

ill with epidemic hepatitis. On subsequent inoculation of the treated tissue the course of the disease in the mice was so modified that Bohmen believes the serum of convalescent patients contains an antibody which destroys the virus. It is also present in variable amounts in the serum of healthy soldiers fighting on the eastern front. Experiments with the serum from 25 children who had never been exposed to epidemic hepatitis were not completed. The virus is thermolabile, being killed at incubator temperatures after 3 to 4 hours, though unaltered after 14 days in the frozen state. (N.B. - The tissues supposedly inactivated by the convalescent serum had also been kept at incubator temperatue for two hours.)

It seems unlikely that this virus is identical with the mouse bronchopneumonia viris of Gonnert, or the strains "lb" and "Greifswald" of Herzberg and Gross. These strains fail to produce any liver lesions, nor can they be transmitted except by intrapulmonary injection.

Kriegsnephritis

Stabsarzt Prof. Randerath:

The Kriegsnephritis or Feldnephritis of this war first appeared in large numbers during the Russian winter campaign of 1941-42. In a single German army there were over 700 cases in the period from 1 Dec 41 to 31 Mar 42. This occurrence of the disease during the winter months duplicates the experiences of World War I when the predisposing factors were recognized as cold, wet, exhaustion, and nutritional deficiency. During this war also the general impression has been gained that cold is the primary factor. This produces disturbances of the sympathetic nervous system which in turn result in impaired blood flow through the skin and kidneys. These, however, are only predisposing factors and the exciting cause is probably an infection, most often streptococcal. The latter, as was shown by Letterer, is usually associated with hypersensitivity of the host. Because of the prevalence and often extraordinarily fulminant course of the disease, the question was raised whether Kriegsnephritis is the same as the nephritis observed in times of peace. During World War I the histopathologic studies of Borst, Aschoff, Löhlein, Rössle, and Herxheimer showed that the anatomical picture of Kriegsnephritis is identical with that of acute glomerulonephritis.

Oberfeldarzt Sponholz:

A total of 355 autopsied cases of Kriegsnephritis were

examined from the viewpoint, so often expressed by clinicians, that this is a particular form of virus infection. All cases in which there existed likelihood of a focal infection, including wounds and frostbite, were therefore excluded. There remained a total of 136 cases which formed the basis of the report. A massive edema of the skin and viscera, particularly of the lungs, was characteristic. The serous effusions showed a marked tendency to secondary infection, offering evidence of the greatly diminished resistance of these patients. The frequency of bronchopneumonia and enteritis may be interpreted in the same way. Grossly the kidneys were of the "large pale" variety found in uncomplicated glomerulonephritis.

A smaller group consisted of those cases that had died suddenly following an illness of less than 24 hours, unaccompanied by any prodromal symptoms, but associated with massive edema and circulatory collapse. At autopsy an acute glomerulonephritis was found; death was due in some instances to uremia, in others to an acute cardiac dilatation. Sudden death in cases of acute nephritis is not peculiar to the Kriegsnephritis, though in the latter it was almost invariably associated with transportation of the patient. In a frightfully high percentage of cases death from nephritis could be directly attributed to the primitive transportation facilities employed on the eastern front.

On histological examination the renal changes are identical with those seen in acute or subacute glomerulonephritis. The space of Bowman's capsule is distended, the glomerular tufts are very cellular as the result of a more or less dense leukocytic infiltrate. At first the capsular epithelium is unaltered, later demilunes appear but they never become prominent. The basic lesion is therefore, an intracapsular glomerulitis which is characterized by its non-progressive character with relative absence of scarring. In a number of cases the duration of the disease covered a period of 3 to 3-1/2 months, yet in these there is no sign of an incipient "shrunken" kidney. During the first 3 to 4 weeks the changes in the tubular epithelium are minimal, later there is evidence of epithelial degeneration with cloudy swelling, some fatty degeneration, and occasional vacuole formation. However, these tubular changes do not bear any relation to the severity of the nephritic syndrome in the clinical course of the disease. The number of erythrocytes in the capsular spaces and tubules is generally small and apparently without relation to the duration or severity of the disease. The so-called "flea bitten" kidneys were seldom seen.

However, there is a particular group of cases in which Kriegsnephritis and typhus fever occur simultaneously. In these there is a diffuse glomerulonephritis of the most pronounced hemorrhagic character. It is apparently without significance, as well as difficult to determine, which disease appeared first. The prognosis of such cases is always grave and they are to be differentiated from the occasional cases of diffuse glomerulonephritis due to typhus. Sponholz believes the latter are quite rare, though Randerath and Herzog claim to have seen them in 1/4 to 2/3 of all cases of typhus fever. The greatly increased permeability of the renal capillaries and diapedesis of erythrocytes in the combined diseases is due to the general vascular damage of typhus combined with the specific renal injury of Kriegsnephritis.

Even though there is nothing in the histopathology of Kriegsnephritis which permits its differentiation from the glomerulonephritis of peacetime, the possibility of a specific infectious agent for the former cannot be ruled out.

Renal "Crush Syndrome"

Repeated questioning of German pathologists failed to reveal any knowledge of the "crush syndrome" of Bywaters. However, in his last quarterly report to the Sanitätsinspekteur, Oberfeldarzt Herzog (Giessen) wrote: "Of interest is another case of burial beneath debris during a bombing raid. There were extensive crush injuries of the body musculature; death occurred after several days with evidence of myoglobinuria. Other similar cases were examined in the pathological institute at Giessen and were reported at the pathology meeting in Breslau by Dr. A. Rothmann in 1944".

Several pathologists stated that the manuscripts of the reports given at this meeting were forwarded to Berlin, but their publication was deferred upon the collapse of German military resistance.

Pulmonary Tuberculosis

There was a definite increase in the incidence of pulmonary tuberculosis in Germany during the war. In 1939 there were 6.3 cases per 10,000 population; in 1942 the incidence of the disease rose to 12-14, and in 1944 it was estimated at 20-22.

Oberstabsarzt H. Wurm (Wiesbaden):

Fatal acute pulmonary tuberculosis of young adults is nearly always associated with widespread dissemination of the infection, and tuberculous meningitis is the immediate cause of death in 72% of the cases. In the Wehrmacht over 50% of the fatal cases of tuberculosis were the result of a primary infection contracted while in the service. There is no fundamental difference between the pathogenesis of the primary pulmonary complex in children and that in young adults. A progressive chronic pulmonary tuberculosis is uncommon in the late primary infection. A generalized form of the disease was more frequent because of the poorer general health of the patient and the prevalence of almost complete physical exhaustion among soldiers on the eastern front. An exudative tuberculous pleurisy often accompanies the progressive late primary complex. Erythema nodosum, frequently observed in the Scandinavian countries, is not seen in Germany.

Histopathological studies have yielded little that is new. Of interest is Wurm's belief that the true Langhans giant cell is the result of abnormal proliferation of a capillary bud. He admits that some of the giant cells in a tubercle are of the foreign body type in which the multiple nuclei are the result of amitotic division.

Coronary Occlusion

The frequency of sudden death associated with coronary occlusion in soldiers under 30 years of age attracted the attention of many pathologists in the Wehrmacht. The most extensive work on this problem was carried out by Oberstabsarzt Büchner and his assistants Meesen and Mueller at the Luftfahrt-Pathologisches Institut at the University of Freiburg. These workers contend that small atheromatous plaques suddenly become very edematous due to a local spasm of the vaso vasora. The resultant anoxemia increases the Hion concentration of the plaque which then absorbs large quantities of globulin from the plasma. It is noteworthy that in the local intimal swellings found at autopsy, little lipoid is demonstrable. The possibility that these changes occur in response to some allergen has also been considered.

In general, the opinion of most German pathologists is that these deaths are associated in some manner with vascular spasms brought about by an "imbalance" in the autonomic nervous system. Whether the spasms can produce death directly, or only indirectly by leading to further structural changes in a previously damaged intima, is the subject of further study. There is apparent unanimity of opinion that the lesions are not identical with the

atherosclerotic plaques found in the coronary vessels of older patients.

Frostbite

During the winter of 1941-42 the troops suffered intensely from cold. Interrogation of men and officers stationed on the eastern front at that time reveals that adequate clothing was not available until the spring of 1942. Not infrequently sentries were found frozen to death at their posts even though the period of duty was short.

Frostbite of the feet and legs was ten times more common than frostbite of the hands and arms. Freezing of the head, buttocks, and genitalia accounted for only 1-2% of all cases. In about 50% the injury was bilateral, i.e. both hands or both feet. Frostbite of first and second degree made up 90-95% of all cases; third and fourth degree injuries comprised the remainder. The following regions were particularly susceptible: Head--the tip of the nose, margin of the ears, ear lobes, and less frequently the scalp. Hands--the 2nd and 5th fingers; the thumb was resistant. Feet--the 1st, 2nd and 5th toes, and the heel. The ankle and patella were also injured.

Oberfeldarzt Prof. Schultz:

Of particular importance to the pathologist in the field are the immediate complications of frostbite, namely, suppurative thrombophlebitis, extensive phlegmons, metastatic suppurative arthritis, and endocarditis (10% Bohne). The nephroses are a questionable sequel of a general lowering of the body temperature (in 50% of cases with 3rd degree frostbite, Bohming).

No specific anatomical cause of death has been noted after overall freezing. However, Dr. Lutz, director of the Forschungstelle für Luftfahrtmedizin at the University of Munich, found that animals cooled below a certain temperature died of anoxemia. This is due to the stability of the oxygen-hemoglobin union at low temperatures, with the result that oxygen is not released in the tissues. On increasing the air pressure to 4 atmospheres, sufficient oxygen is carried by the plasma to support the lowered metabolism of the animal at the reduced temperatures.

Also of importance in general lowering of the body

temperature is displacement of the blood mass to the internal organs. The weight of the liver is increased, perhaps the result of "cloudy swelling". According to Lucke there is an absence of glycogen in the liver and skeletal muscles. Schultz found waxy degeneration of the musculature in 37% of all autopsies performed on the eastern front during the winter, as compared with 4% during the summer. Changes in the adrenal gland are also seen but have not yet been evaluated.

Stabsarzt Prof. Lundle:

Lundle investigated the effect of extreme cold on the rat's tail and rabbit's ear. The capillary system suffered greatest injury and was manifested by blood stasis, edema, faulty nutrition of the tissues, blister formation, etc. Puncture of the vessels in a thawing ear revealed that the blood was hemolysed, and that during hemolysis vasodilator substances are formed. A histamin-like substance was isolated. When the frozen ears were transfused with a tellurite solution there was evidence of injury to the oxidation-reduction mechanism, although the adrenals still reacted to adrenalin.

Oberstabsarzt Prof. Staemmler:

Vascular Injury. In the zone of demarcation an endangiitis is frequently observed. This is due to 1) extension of an inflammatory process in the adventitia to the intima, and 2) plasma infiltration of the intima. Endophlebitis with xanthomatous, fat containing granulomata was occasionally noted in the veins. The relation of the time lapse since freezing and the age of the vascular changes indicates that such changes may progress for some time after the initial injury. Progressive endarteritis may ultimately lead to gangrene of the part.

Nerve Injury. These are very common and may be severe. They are manifested by a diffuse destruction of the myelin sheath, damage to the axis cylinders, and an accumulation of fat droplets and foam cells. The lesion may extend far beyond the limits of the tissue that was immediately affected by frostbite. An intense perineuritis and endo-neuritis which is not dependent upon the severity of the other changes in the nerve is often observed.

Injuries to Other Tissues. Atrophy and hyperkera-tosis of the epidermis, damage to the elastic fibers of the corium, and vascular degeneration of the sweat glands are common. Atrophy of bone and muscle occurs even out-side the actual area of injury. Dr. Wurm (Wiesbaden) found a diffuse interstitial myocarditis identical with

that of diphtheria in 12 cases of frostbite followed by gangrene of the extremities. The lesions were similar to those found following extensive burns; numerous cultures were negative for C. diphtheriae.

Oberstabsarzt Prof. Siegmund (Muenster):

Impaired blood flow through the skin of a region subjected to prolonged cold is recognized by pallor of the part, and is the first sign of injury due to cold. This ischemia, the result of reflex constriction of the arterioles, persists until increasing warmth of the environment stimulates the heat receptors, leading to vascular dilatation.

The fate of tissues subjected to cold is dependent upon the duration of the accompanying ischemia, rather than upon the intensity of the cold. After the oxygen is exhausted and carbon dioxide has accumulated, tissue respiration switches to aerobic glycolysis and fermentation. Lactic acid accumulates, and the oxidative deaminization of the amino acids is disturbed, resulting in the formation of amines that act as vasodilators. When glycolysis ceases the tissues die. The precipitation of tissue colloids and the accumulation of the acid by-products of metabolism in the cells, leads to an increase in their osmotic pressure with resultant swelling. This swelling offers a purely physical explanation for the increased permeability of cell membranes in the ischemic area. Changes in the electrolyte concentration accompanies the decreased electrical potential of the plasma proteins, thus providing the conditions for an agglutination of the erythrocytes.

If blood flow is reestablished at an early date there will be a local hyperemia (due to the vasodilator effect of locally accumulated amines?) and only a small transudation of plasma proteins into the tissues. This is quickly removed by the lymphatics and a normal circulation is restored. If the transudate accumulates more rapidly, blisters will develop which are filled with clear yellow fluid. These changes are characteristic of a 1st degree frostbite.

After more severe injury due to cold and anoxemia, normal tissue metabolism is not resumed, even when the arterial circulation has been reestablished. The blood vessels are maximally distended by the inflowing blood, but the plasma proteins pass through the capillary walls in great quantities. Under the abnormal conditions

existing in the tissues the plasma penetrates the cell membrances, leading to karyolysis, chromatolysis, and cell death. Simultaneously, the erythrocytes agglutinate, the plasma proteins are precipitated in the dilated vessels, and hemolysis rapidly takes place. The endothelial cells of the proximal and less severely injured portions of the vessels proliferate and cover the thrombus. The thrombus subsequently undergoes organization.

These changes are irreversible and lead to death of the tissue with the signs and symptoms of dry gangrene. The reddish-black discoloration of the region is due to the distension of its capillaries and larger vessels by the agglutinated erythrocytes, and the effusion of the breakdown products of hemoglobin into the surrounding tissues. The condition is recognized clinically as a 3rd degree frostbite.

SUMMARY

The plan of organization for the conduct of pathology in the Wehrmacht resembled that of the U. S. Army. The chief pathologist, attached to the Sanitätsinspektion, was also director of the Institut für Allgemeine Wehrpathologie in Berlin. This institute, however, served mainly as a repository for the protocols of autopsies performed in the Wehrmacht. Its diagnostic and research activities were very limited.

A pathologist was attached to each Wehrkreis and large general hospital in the zone of the interior, and to each army group, army, and major general hospital in the theater of operations. The pathologists of the Luftwaffe formed a separate organization. It was headed by Oberstabsarzt Büchner, director of the Luftfahrt-Pathologisches Institut at the University of Freiburg.

The larger establishments were housed in civilian laboratories in Germany or in the occupied countries. A field unit, consisting of three chests, provided an adequate work bench, as well as equipment for the conduct of histopathological studies of the performance of postmortem examinations.

The Russian campaign provided much of the material for special pathological investigations. Most important of these were the studies on typhus, epidemic hepatitis, acute glomerulonephritis (Kriegsnephritis), and frostbite. Also investigated were Wolhynian (trench) fever, bacillary

and amoebic dysentery, malaria, diphtheria, tularemia, tuberculosis, and coronary occlusion. No epidemics or unusual disease entities appeared among the civilian population following the intensive Allied air attacks upon the cities. The renal lesions that follow extensive crushing injuries, first noted by Bywaters during the London blitz, were studied by only a few German pathologists.

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